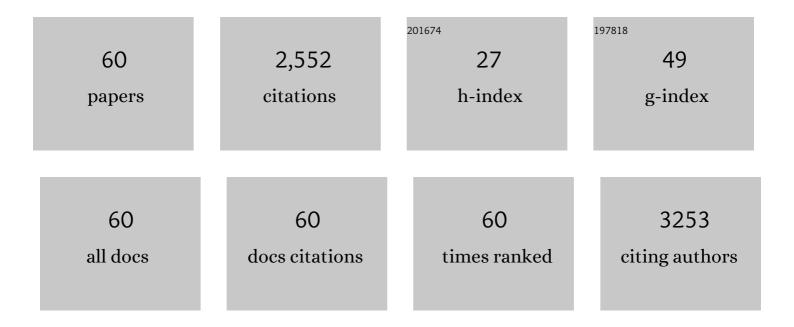
Tomoko Takano

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	The Canadian Glomerulonephritis Registry (CGNR) and Translational Research Initiative: Rationale and Clinical Research Protocol. Canadian Journal of Kidney Health and Disease, 2022, 9, 205435812210890.	1.1	1
2	Rho GTPase regulatory proteins in podocytes. Kidney International, 2021, 99, 336-345.	5.2	26
3	Role of Rho GTPase Interacting Proteins in Subcellular Compartments of Podocytes. International Journal of Molecular Sciences, 2021, 22, 3656.	4.1	10
4	Mammalian target of rapamycin is activated in the kidneys of patients with scleroderma renal crisis. Journal of Scleroderma and Related Disorders, 2020, 5, 152-158.	1.7	2
5	ARHGEF7 (β-PIX) Is Required for the Maintenance of Podocyte Architecture and Clomerular Function. Journal of the American Society of Nephrology: JASN, 2020, 31, 996-1008.	6.1	12
6	Multivalent nephrin/Nck interactions define a threshold for clustering and tyrosine-dependent nephrin endocytosis. Journal of Cell Science, 2020, 133, .	2.0	11
7	Salt Sensing by Serum/Glucocorticoid-Regulated Kinase 1 Promotes Th17-like Inflammatory Adaptation of Foxp3+ Regulatory T Cells. Cell Reports, 2020, 30, 1515-1529.e4.	6.4	33
8	Targeting the mTOR pathway uncouples the efficacy and toxicity of PD-1 blockade in renal transplantation. Nature Communications, 2019, 10, 4712.	12.8	76
9	From podocyte biology to novel cures for glomerular disease. Kidney International, 2019, 96, 850-861.	5.2	49
10	The Canadian childhood nephrotic syndrome (CHILDNEPH) study: report on mid-study feasibility, recruitment and main measures. BMC Nephrology, 2019, 20, 159.	1.8	4
11	Recessive mutation in CD2AP causes focal segmental glomerulosclerosis in humans and mice. Kidney International, 2019, 95, 57-61.	5.2	11
12	ShcA Adaptor Protein Promotes Nephrin Endocytosis and Is Upregulated in Proteinuric Nephropathies. Journal of the American Society of Nephrology: JASN, 2018, 29, 92-103.	6.1	24
13	The Role of Trio, a Rho Guanine Nucleotide Exchange Factor, in Glomerular Podocytes. International Journal of Molecular Sciences, 2018, 19, 479.	4.1	11
14	Binding and inhibition of the ternary complex factor Elk-4/Sap1 by the adapter protein Dok-4. Biochemical Journal, 2017, 474, 1509-1528.	3.7	3
15	Changing Paradigms in the Management of Rejection in Kidney Transplantation. Canadian Journal of Kidney Health and Disease, 2017, 4, 205435811668822.	1.1	10
16	Rituximab in Minimal Change Disease. Canadian Journal of Kidney Health and Disease, 2017, 4, 205435811769866.	1.1	16
17	Rac1 activation in podocytes induces the spectrumÂof nephrotic syndrome. Kidney International, 2017, 92, 349-364.	5.2	53
18	A Point Mutation in p190A RhoGAP Affects Ciliogenesis and Leads to Glomerulocystic Kidney Defects. PLoS Genetics, 2016, 12, e1005785.	3.5	21

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19	Genetic Ablation of Calcium-independent Phospholipase A2Î ³ Induces Glomerular Injury in Mice. Journal of Biological Chemistry, 2016, 291, 14468-14482.	3.4	19
20	Nephrin Tyrosine Phosphorylation Is Required to Stabilize and Restore Podocyte Foot Process Architecture. Journal of the American Society of Nephrology: JASN, 2016, 27, 2422-2435.	6.1	65
21	Disease-causing mutations of RhoGDIα induce Rac1 hyperactivation in podocytes. Small GTPases, 2016, 7, 107-121.	1.6	22
22	Apoptosis inhibitor of macrophage protein enhances intraluminal debris clearance and ameliorates acute kidney injury in mice. Nature Medicine, 2016, 22, 183-193.	30.7	161
23	Loss of Rho-GDlα sensitizes podocytes to lipopolysaccharide-mediated injury. American Journal of Physiology - Renal Physiology, 2015, 308, F1207-F1216.	2.7	6
24	Calcium-independent Phospholipase A2γ Enhances Activation of the ATF6 Transcription Factor during Endoplasmic Reticulum Stress. Journal of Biological Chemistry, 2015, 290, 3009-3020.	3.4	13
25	Rho-GTPase Signalling in the Pathogenesis of Nephrotic Syndrome. Advances in Nephrology, 2014, 2014, 1-11.	0.2	5
26	Role of Guanine Nucleotide Exchange Factor-H1 in Complement-mediated RhoA Activation in Glomerular Epithelial Cells. Journal of Biological Chemistry, 2014, 289, 4206-4218.	3.4	10
27	Complement-mediated glomerular injury is reduced by inhibition of protein-tyrosine phosphatase 1B. American Journal of Physiology - Renal Physiology, 2014, 307, F634-F647.	2.7	9
28	Protein Tyrosine Phosphatase 1B Inhibition Protects against Podocyte Injury and Proteinuria. American Journal of Pathology, 2014, 184, 2211-2224.	3.8	28
29	Role of Rho-GTPases and their regulatory proteins in glomerular podocyte function. Canadian Journal of Physiology and Pharmacology, 2013, 91, 773-782.	1.4	48
30	Complement-Mediated Cellular Injury. Seminars in Nephrology, 2013, 33, 586-601.	1.6	60
31	<i>ARHGDIA</i> : a novel gene implicated in nephrotic syndrome. Journal of Medical Genetics, 2013, 50, 330-338.	3.2	92
32	Complement-mediated Activation of Calcium-independent Phospholipase A2γ. Journal of Biological Chemistry, 2013, 288, 3871-3885.	3.4	28
33	Nuclear factor of activated T cells mediates RhoA-induced fibronectin upregulation in glomerular podocytes. American Journal of Physiology - Renal Physiology, 2013, 304, F849-F862.	2.7	12
34	Pathogenesis of common glomerular diseases – role of the podocyte cytoskeleton. Cell Health and Cytoskeleton, 2012, , 103.	0.7	2
35	Podocyte Protein, Nephrin, Is a Substrate of Protein Tyrosine Phosphatase 1B. Journal of Signal Transduction, 2011, 2011, 1-10.	2.0	30
36	Role of the retinoic acid receptor-α in HIV-associated nephropathy. Kidney International, 2011, 79, 624-634.	5.2	64

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37	Activation of RhoA in Podocytes Induces Focal Segmental Glomerulosclerosis. Journal of the American Society of Nephrology: JASN, 2011, 22, 1621-1630.	6.1	114
38	Endoplasmic reticulum stress in glomerular epithelial cell injury. American Journal of Physiology - Renal Physiology, 2011, 301, F496-F508.	2.7	21
39	Cytosolic phospholipase A2-α enhances induction of endoplasmic reticulum stress. Biochimica Et Biophysica Acta - Molecular Cell Research, 2010, 1803, 468-481.	4.1	12
40	Rac1 Contributes to Actin Organization in Glomerular Podocytes. Nephron Experimental Nephrology, 2010, 114, e93-e106.	2.2	33
41	p21-Activated kinases regulate actin remodeling in glomerular podocytes. American Journal of Physiology - Renal Physiology, 2010, 298, F951-F961.	2.7	26
42	Podocyte Injury and Albuminuria in Mice with Podocyte-Specific Overexpression of the Ste20-Like Kinase, SLK. American Journal of Pathology, 2010, 177, 2290-2299.	3.8	18
43	Nck Proteins Maintain the Adult Glomerular Filtration Barrier. Journal of the American Society of Nephrology: JASN, 2009, 20, 1533-1543.	6.1	81
44	The cytoprotective role of Ras in complement-mediated glomerular epithelial cell injury. Clinical Immunology, 2009, 131, 343-353.	3.2	4
45	Role of calcium-independent phospholipase A ₂ in complement-mediated glomerular epithelial cell injury. American Journal of Physiology - Renal Physiology, 2008, 294, F469-F479.	2.7	20
46	Role of Rho-GTPases in complement-mediated glomerular epithelial cell injury. American Journal of Physiology - Renal Physiology, 2007, 293, F148-F156.	2.7	32
47	Rat nephrin modulates cell morphology via the adaptor protein Nck. Biochemical and Biophysical Research Communications, 2006, 349, 310-316.	2.1	44
48	Nck adaptor proteins link nephrin to the actin cytoskeleton of kidney podocytes. Nature, 2006, 440, 818-823.	27.8	432
49	Prostaglandin E2 promotes cell survival of glomerular epithelial cells via the EP4 receptor. American Journal of Physiology - Renal Physiology, 2006, 290, F1534-F1542.	2.7	47
50	Role of the Endoplasmic Reticulum Unfolded Protein Response in Glomerular Epithelial Cell Injury. Journal of Biological Chemistry, 2005, 280, 24396-24403.	3.4	101
51	The actin cytoskeleton facilitates complement-mediated activation of cytosolic phospholipase A2. American Journal of Physiology - Renal Physiology, 2004, 286, F466-F476.	2.7	18
52	Src-Family Kinase Fyn Phosphorylates the Cytoplasmic Domain of Nephrin and Modulates Its Interaction with Podocin. Journal of the American Society of Nephrology: JASN, 2004, 15, 3006-3015.	6.1	116
53	Inhibition of Cyclooxygenases Reduces Complement-Induced Glomerular Epithelial Cell Injury and Proteinuria in Passive Heymann Nephritis. Journal of Pharmacology and Experimental Therapeutics, 2003, 305, 240-249.	2.5	37
54	p38 Mitogen-activated protein kinase protects glomerular epithelial cells from complement-mediated cell injury. American Journal of Physiology - Renal Physiology, 2003, 285, F765-F774.	2.7	47

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55	Complement Activates the c-Jun N-Terminal Kinase/Stress-Activated Protein Kinase in Glomerular Epithelial Cells. Journal of Immunology, 2002, 169, 2594-2601.	0.8	55
56	Complement C5b-9 Membrane Attack Complex Increases Expression of Endoplasmic Reticulum Stress Proteins in Glomerular Epithelial Cells. Journal of Biological Chemistry, 2002, 277, 41342-41351.	3.4	107
57	Complement C5b-9 induces cyclooxygenase-2 gene transcription in glomerular epithelial cells. American Journal of Physiology - Renal Physiology, 2001, 281, F841-F850.	2.7	21
58	Complement C5b-9 induces cyclooxygenase-2 gene transcription in glomerular epithelial cells. American Journal of Physiology - Renal Physiology, 2001, 281, F841-F850.	2.7	10
59	Complement-induced phospholipase A2 activation in experimental membranous nephropathy1 See Editorial by Shankland, p. 1204 Kidney International, 2000, 57, 1052-1062.	5.2	45
60	Complement C5b-9-Mediated Arachidonic Acid Metabolism in Glomerular Epithelial Cells. American Journal of Pathology, 2000, 156, 2091-2101.	3.8	64